VII-B. PULMONARY CIRCULATION

The pulmonary vascular lesions in pulmonary emphysema have been summarized by Liebow (1959). The lesions in the small pulmonary arteries are severe enough to cause elevation of pulmonary arterial blood pressure (Hicken et al., 1965). Electron microscopy has revealed damage to the endothelium, filling of the capillary lumina with collagen, and recanalization of the damaged vessels (Martin and Boatman, 1965). Crenshaw (1968) has reviewed the hypothesis that pulmonary emphysema has a vascular etiology. However, none of the vascular lesions seen in human pulmonary emphysema have been reproduced in animals by cigarette smoking. The lungs of healthy cigarette smokers show fibrous thickening which is also seen in the aging lungs of nonsmokers (Auerbach et al., 1968).

The acute effect of smoking one cigarette was studied in 67 Indian patients suffering from chronic bronchopulmonary disease (Viswanathan, 1965). There was a significant rise in mean and diastolic pressure in the pulmonary artery, but no significant change in systolic. The change in diastolic pressure was interpreted to mean vasoconstriction, although there was no measurement of cardiac output nor of left atrial pressure to allow estimation of pulmonary vascular resistance. The fall in pulmonary compliance seen among smokers does not relate to changes in the pulmonary circulation (Krumholz et al., 1965).

There are some observations relating to pulmonary circulation in animals exposed to cigarette smoking. Larson et al. (1965) used nicotine intravenously and noted a biphasic rise and then fall in pulmonary vascular resistance in the dog. Samanek and Aviado (1966) noticed a rise in pulmonary arterial blood pressure following inhalation of cigarette smoke, but the

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response could not be repeated in the same animal for purposes of eliciting the mechanism of the rise. Nicotine injection was utilized and the following mechanisms were identified: vasoconstriction by release of catecholamine stores in the lung and the release of histamine.

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VII-C. CARDIAC FUNCTION

Since cigarette smoking has been identified as the most important cause of pulmonary emphysema by the United States Public Health Service, it has been implicated in cor pulmonale (Parade, 1969; Behnke et al., 1970). In a group of 150 patients with cor pulmonale in India, 28% were nonsmokers (Sanghvi and Kotia, 1963). In another group of 134 patients in India, 39% were nonsmokers (Khosla et al., 1969). In the United States the proportion of nonsmokers is lower among patients with cor pulmonale; Cullen et al. (1970) reported from New York only 3 out of 60 patients. Patients dying of cor pulmona have manifested failure of the left ventricle but the role of cigarette smoking has not been proven (Mitchell et al., 1968; Rao et al., 1969; Strong et al., 1969).

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VII- D. PULMONARY SURFACTANT AND OTHER CONSTITUENTS

Stability of the pulmonary alveoli is partially due to changes in surface tension. The surface active component of lung extract which is responsible for maintaining alveolar distension has been called surfactant. Bondurant (1960), Miller et al (1960) and Miller and Bondurant (1962) reported that the application of cigarette smoke to lung extract caused a decrease in surface tension. They also reported that lung extract prepared from rats which have been exposed to smoke in life have lower surface tension than that of unexposed rats. They concluded that these results may relate to the pathogenesis of pulmonary emphysema.

Subsequent reports have confirmed the reduction of surfactant in bronchial washings of the dog by in vitro application (Webb et al., 1967) and in the dog, cat and guinea pig by in vivo inhalation of cigarette smoke (Giammona, 1967; Sekulic et al., 1968). Two recent reports include the incorporation of radioactive labeled palmitic acid into pulmonary surfactant. Giammona et al. (1971) used unanesthetized dogs exposed repeatedly to cigarette smoke daily for 37 to 87 days. They noted no change in surface activity or in rate of time of incorporation of palmitic acid in these dogs. Balint et al. (1971) used anesthetized dogs and administered cigarette smoke by positive ventilation from 90 to 220 min. They reported a decrease in choline and phosphate incorporation in the lecithin of the lung. The results in the unanthestetized dog are similar to the effects obtained by the usual use of tobacco in man, whereas those in the anesthetized dog are toxicological in nature.

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Measurement of surfactant activity in man has been made on bronchial washings. Cook and Webb (1966) noted a decrease of surfactant action in nonsmokers with lung disease and in chronic smokers with or without demonstrable pulmonary disease. A reduction in the white layer of the lavage fluid after centrifugation has been reported in smokers (Pratt et al., 1969). There is a deficit in total lipid content, particularly of lecithin, in the lavage from smokers as compared with that from nonsmokers (Finley and Ladman, 1972). These observations have been commented upon by Clements (1972), Sachs (1972) and Finley (1972) in relation to the shortcomings of using lavage fluid in interpreting the events occurring in the alveolar lining.

There are other reported changes in the lung of patients with chronic lung disease. The reduction in tissue content of cadmium and zinc in patients with chronic bronchitis or pulmonary emphysema by Szadkowski et al. (1969). Lewis et al. (1969), and Morgan (1971) have not been examined in the lungs of smokers without lung disease. Inhalation of cigarette smoke causes a release of histamine from lung tissue (Aviado et al., 1966) and from the guinea pig lung (Saindelle et al., 1968; Lewis and Nicholls, 1972). Exposure to cigarette smoke causes an increase in protein-mucopolysaccharide complexes in the connective tissue of the guinea pig lung (Lupu and Velican, 1962), an increase in hydroxyproline content of the mouse lung (Rosenkrantz et al., 1969) and a change in electron spin resonance of the rabbit lung (Rowlands et al., 1967). The significance of these observations in relation to lung disease has not been elucidated.

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VIII. COMMENTARY ON SELECTED ARTICLES AND THEIR PUBLICITY

VIII-A. COMMENTARY ON U.S. PUBLIC HEALTH SERVICE PUBLICATIONS

The author of this review has prepared a page by page critique of the seven documents distributed by the U.S. Public Health Service. A few examples of misquotation, omission and misinterpretation have been selected and are appended in this report. The reports have been identified by the year of their appearance. The pages pertinent in the subject of pulmonary disease are as follows:

		Reprint
(1)	1964: Smoking and Health. pp 259-315.	810
(2)	1967: The Health Consequences of Smoking. pp 87-124.	811
(3)	1968: The Health Consequences of Smoking. pp 63-86.	812
(4)	1969: The Health Consequences of Smoking. pp 35-52.	813
(5)	1971: The Health Consequences of Smoking. pp 137-230.	814
(6)	1972: The Health Consequences of Smoking. pp 35-56.	815
(7)	1973: The Health Consequences of Smoking. pp 43-91.	816

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1. Change in conclusions regarding the relationship of cigarette smoking to pulmonary emphysema and chronic bronchitis (Reports 1964, 1967 and 1971).

CONCLUSIONS OF THE 1964 REPORT (59)

1. Cigarette smoking is the most important of the causes of bronchitis in the United States, and increases the risk of dying from chronic bronchitis.

2. A relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal. The smoking of cigarettes is associated with an increased risk of dying from pulmonary emphysema.

3. For the bulk of the population of the United States, the importance of cigarette smoking as a cause of chronic bronchopulmonary disease is much greater than that of atmospheric pollution or occupational exposures.

4. Cough, sputum production, or the two combined are consistently more frequent among cigarette smokers than among nonsmokers.

5. Cigarette smoking is associated with a reduction in ventilatory function. Among males, cigarette smokers have a greater prevalence of breathlessness than nonsmokers.

6. Cigarette smoking does not appear to cause asthma.

7. Although death certification shows that cigarette smokers have a moderately increased risk of death from influenza and pneumonia, an association of cigarette smoking and infectious diseases is not otherwise substantiated.

HIGHLIGHTS OF THE 1967 REPORT (57)

1. New data confirm and to some extent strengthen the conclusions of the Surgeon General's 1964 Report.

2. Cigarette smoking is the most important of the causes of chronic non-neoplastic bronchopulmonary diseases in the United States. It greatly increases the risk of dying not only from both chronic bronchitis but also from pulmonary emphysema.

3. Cessation of smoking is followed by a reduction in mortality from chronic bronchopulmonary disease relative to the mortality of those who continue to smoke.

4. Even relatively young cigarette smokers frequently have demonstrable respiratory symptoms and reduction in ventilatory function.

1971 SUMMARY AND CONCLUSIONS

1. Cigarette smoking is the most important cause of chronic obstructive bronchopulmonary disease in the United States. Cigarette smoking increases the risk of dying from pulmonary emphysema and chronic bronchitis. Cigarette smokers show an increased prevalence of respiratory symptoms, including cough, sputum production, and breathlessness, when compared with nonsmokers. Ventilatory function is decreased in smokers when compared with nonsmokers.

2. Cigarette smoking does not appear to be related to death from bronchial asthma although it may increase the frequency and severity of asthmatic attacks in patients already suffering from this disease.

3. The risk of developing or dying from COPD among pipe and/or cigar smokers is probably higher than that among nonsmokers while clearly less than that among cigarette smokers.

4. Ex-cigarette smokers have lower death rates from COPD than do continuing smokers. The cessation of cigarette smoking is associated with improvement in ventilatory function and with a decrease in pulmonary symptom prevalence.

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5. Young, relatively asymptomatic, cigarette smokers show measurably altered ventilatory function when compared with nonsmokers of the same age.

6. For the bulk of the population of the United States, the importance of cigarette smoking as a cause of COPD is much greater than that of atmospheric pollution or occupational exposure. However, exposure to excessive atmospheric pollution or dusty occupational materials, and cigarette smoking may act jointly to produce greater COPD morbidity and mortality.

7. The results of experiments in both animals and humans have demonstrated that the inhalation of cigarette smoke is associated with acute and chronic changes in ventilatory function and pulmonary histology. Cigarette smoking has been shown to alter the mechanism of pulmonary clearance and adversely affect ciliary function.

8. Pathological studies have shown that cigarette smokers who die of diseases other than COPD have histologic changes characteristic of COPD in the bronchial tree and pulmonary parenchyma more frequently than do nonsmokers.

9. Respiratory infections are more prevalent and severe among cigarette smokers, particularly heavy smokers, than among nonsmokers.

10. Cigarette smokers appear to develop postoperative pulmonary complications more frequently than nonsmokers.

In the 1964 report, cigarette smoking is "the most important of the causes of chronic bronchitis" and for pulmonary emphysema "it has not been established that the relationship is causal."

By 1967, the two conditions were combined and cigarette smoking becomes "the most important of the causes of chronic non-neoplastic bronchopulmonary diseases."

In the latest summary, which appeared in 1971, cigarette smoking is identified as "the most important cause of chronic obstructive bronchopulmonary disease."

The change in terminology should be noted - from a separate identification of chronic bronchitis and pulmonary emphysema, finally to the all-inclusive name of chronic obstructive bronchopulmonary disease. This last term is used also for morbidity and mortality statistics, although death rates for pulmonary emphysema as distinct from chronic bronchitis are available. The reviewer is now in the process of evaluating the significance of the prevalence of bronchitis and emphysema separately.

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2. Experimental proof that cigarette smoking produces pulmonary emphysema in animals. The 1967 and 1971 reports contain an adequate coverage of Auerbach's experiments.

1967 Auerbach and his associates (*5, 6*) tracheostomized 10 adult beagles and, in an attempt to approximate human smoking more closely, exposed them to cigarette smoke through the tracheostomy tube. Five dogs died during this experiment and the remaining five were sacrificed after approximately 14 months of exposure. Other beagles were kept as controls; two had tracheostomy openings. These control dogs were sacrificed at the time the last five smoking dogs were sacrificed. Lungs of the dogs exposed to cigarette smoke showed microscopically the presence of dilated air spaces, especially beneath the pleural surface. Here the alveolar septa showed a fibrous thickening of the walls with areas of rupture and dilated air sacs. Padlike attachments to alveolar septa were found. These zones of connective tissue surrounding dilated air sacs were also visible macroscopically as white areas on the lung surface. There was no thickening of the walls of small arteries and arterioles within the lung. The lungs of the control dogs were normal in appearance with none of these changes. These abnormalities approximate but are not fully concordant with some of the typical pathological findings in human emphysema. This experiment does indicate that inhaled cigarette smoke apparently can damage the pulmonary parenchyma of dogs. Other findings (*6*) as yet unpublished, indicate that abnormalities of the bronchial epithelium resulted that approximate many of the histopathologic findings of human chronic bronchitis.

A71 The investigations of Auerbach and his coworkers (*15, 16, 88*) have demonstrated by the use of both light and electron microscopy that dogs who inhale cigarette smoke through tracheostomas develop progressively more severe lesions of the bronchi and parenchyma with increased exposure to cigarette smoke. In electron microscopic studies of specimens taken from the lungs of dogs thus exposed to cigarette smoke, the following changes were observed: In 5 dogs sacrificed after only 44 days of smoking exposure, there was a proliferation of goblet cells as well as a partial loss of cilia in the lining cells, and in 5 dogs sacrificed after 420 days or more of exposure, the number of cell layers in the bronchial epithelium was found to be twice that of the nonsmoking dogs. Goblet cells and ciliated columnar cells were no longer present; instead, the surface was lined with columnar and cuboidal cells with stubby projections in place of cilia. Mitotic figures were frequently observed in the basal cells. These findings may be relevant to carcinogenesis as well as to the development of COPD.

In a long-term experiment, carried out by the same group, dogs were exposed to varying doses of cigarette smoke. Details of the experimental procedure have been outlined in the section on Pulmonary Carcinogenesis. The animals were separated into nonsmoker, filter-tip cigarette, nonfilter-light, and nonfilter-heavy exposure groups. The dogs were "smoked" for 875 days, or approximately 29 months. The animals which died during the experiment and the animals sacrificed after day 875 were examined for pulmonary parenchymal changes as well as for bronchial epithelial alterations. As seen in figures 1 and 2, dose-related pathological changes, including fibrosis and emphysema, were found in the lung parenchyma of the exposed dogs. These changes were similar to those seen in the lungs of humans with COPD.

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The experiments which failed to show that cigarette smoking does not produce pulmonary emphysema have not been mentioned in the reports. Some of the work from one laboratory has been quoted to describe details that do not relate to pulmonary emphysema directly:

1968 Aviado and his co-workers (*2, 3, 4, 19, 38, 45, 46, 47, 48*) have continued their studies on bronchoconstriction and bronchodilation in animals and recently have further investigated the role of histamine in a study of inhibitors for histamine decarboxylase in rabbits, dogs, and cats (*39*). These species have variations in response to cigarette smoking as previously noted. Cats have a uniphasic bronchoconstrictor response to inhaled cigarette smoke (somewhat like man's) and dogs have a biphasic response. Rabbits were observed to behave differently than cats or dogs. Histamine has been implicated as mediating part of the bronchoconstrictive effect of cigarette smoke. The rabbit does not respond to histamine by bronchoconstriction. This study (*39*) suggests that the rabbit lacks a histamine sensitive system in the airways, in contrast to cats and dogs. Alpha-hydrazino histidine, which inhibits the enzyme histamine decarboxylase, was demonstrated to prevent much of the bronchoconstrictive effect in cats and dogs. By analogy, this suggests the possibility that histamine may mediate some of the bronchoconstrictive response to inhaled tobacco smoke noted in humans. Pretreatment with atropine has been shown to block the bronchoconstriction caused by cigarette smoke (*30*) and by histamine inhalation in humans (*7, 8, 52*).

1971 Aviado and coworkers have performed a series of experiments on live animals and in heart-lung preparations to study the effect of cigarette smoke on pulmonary physiology and structure (*18, 19, 20, 21, 22, 179, 180, 199, 200, 201, 202*). The authors observed that cigarette smoke causes acute bronchoconstriction both by the release of histamine and the stimulation of parasympathetic nerve pathways in the lung. Bronchial arterial injections of nicotine were found to cause reactions similar to those observed after cigarette smoke inhalation. The bronchoconstriction was usually followed by bronchodilatation which the authors attributed to sympathetic stimulation. As mentioned in the Chapter on Cardiovascular Diseases, nicotine has been shown to induce the release of catecholamines.

Experiments by Aviado and coworkers as well as other authors (*66, 99*) using guinea pigs showed that exposure to cigarette smoke was associated with increased bronchopulmonary resistance and decreased pulmonary compliance. The authors related these changes to the bronchoconstriction of terminal ventilatory units.

Similar experiments in dogs showed that the increase in resistance following either cigarette smoke exposure or intravenous nicotine could be blocked by pretreatment with atropine. As a parasympathetic blocker, atropine would decrease the acute bronchoconstrictive phase.

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Most recently, Aviado and his colleagues (*20, 130*) have attempted to induce physiologic and anatomic changes similar to those found in the lungs of patients with emphysema. They exposed male rats to cigarette smoke, the introduction of the enzyme papain, as well as to partial tracheal ligation. In 10 rats exposed to cigarette smoke twice daily for 30 minutes over a period of 10 weeks, no changes in pulmonary compliance or resistance were noted. Also, no abnormal histological changes were observed in the group exposed only to cigarette smoke. However, animals who underwent tracheal ligation as well as smoke exposure showed increased numbers of enlarged air spaces and increased pulmonary resistance when compared with animals who underwent only tracheal ligation.

1973-Aviado and coworkers have studied the effects of hormones on the pulmonary response to cigarette smoke inhalation and intravenous nicotine injection. Subcutaneous progesterone administration, prior to nicotine or smoke exposure, reduced the bronchoconstrictor response in rats (*64*). A similar experiment involving pretreatment of dogs with glucocorticoids resulted in variable bronchoconstrictor responses after exposure to cigarette smoke (*12*).

In the 1971 quotation, there is no mention that the attempt to induce emphysema in rats by exposure to cigarette smoke failed, although other procedures caused the lesions. The failure to elicit emphysema appeared in the conclusions of two articles which the documents overlooked.

Summary of Ito and Aviado (1968)

Rats exposed to cigarette smoke daily for ten weeks did not develop signs of pulmonary emphysema. The rats subjected to experimental ligation of the trachea and intratracheal injection of papain developed signs of pulmonary emphysema but exposure to cigarette smoke did not uniformly influence the various signs. Functional residual capacity was not markedly elevated although histological examination showed a higher percentage of air spaces in emphysematous rats exposed to cigarette smoke. Cigarette smoke caused an increase in pulmonary resistance only in the rats with experimental emphysema. The increase in resistance is interpreted to mean an increase in bronchomotor tone. Isoproterenol injection induces bronchodilatation, more intensely in rats exposed to cigarette smoke.

Summary of Aviado et al. (1970)

This is the second reported attempt to demonstrate that cigarette smoke can induce pulmonary emphysema in rats. The negative results have been scrutinized to determine the significance of the changes in airway resistance and content of biogenic amines during exposure to cigarette smoke. These changes in the lung are not important in determining the appearance or disappearance of pulmonary emphysema in rats. In the same animal species, tracheal ligation and endotracheal injection of papain produce pulmonary emphysema.

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3. Evaluation of air pollution. The U.S. Public Health documents have arrived at the conclusion that cigarette smoking is the most important factor in the causation of chronic bronchitis and pulmonary emphysema. They have interpreted articles relating to air pollution and industrial hazards as indicating that these are less important than cigarette smoking. A re-examination of 48 articles indicates just the opposite conclusion made by the authors and details are outlined in Sections III and IV. One example is the report of Phelps and Koike (1962). The 1964 report has the following citation:

> In the "Tokyo-Yokohama Asthma" studies, a severe asthma-like disease, presumed to be caused by air pollution, affected cigarette smokers predominantly (155). The absence of smoking data on unaffected members of the same population leaves the question of an additive effect of cigarette smoking unanswered. One study suggests that non-smokers may have a slightly greater prevalence of asthma than smokers; the possibility of bias due to self-selection of the base population could not, however, be excluded in this study (84).

Phelps and Koike concluded that air pollution is the cause of the disability. Removing the patients from the area caused a dramatic relief. The quotations are as follows:

> As the disease progresses (usually in the patient with symptoms who remains in this area for several years or who returns to the area after having had the disease during a previous tour), more shortness of breath is manifested. The patient by this time is usually receiving adrenal steroids and has been frequently hospitalized. It is at this time that all of the symptoms and signs of emphysema are present. This is more likely to occur in persons who are heavy cigarette smokers. Although the study is by no means complete, it is believed that there is evidence that air pollution, of unknown content, is the cause of much disability which, under certain conditions, may progress rapidly to emphysema.
>
> The follow-up studies revealed that those patients who were transferred to the United States early in the course of the disease had dramatic relief of their symptoms. All pulmonary function studies returned to normal within one to two months after the arrival of these patients in the United States. Their function studies took longer to return to normal than their symptoms. Those persons who had their disease for a long period of time (two to three years) took much longer to become asymptomatic, and it took even longer for their pulmonary function studies to become normal. Most of the patients were improved so far as coughing and wheezing were concerned, but they still had shortness of breath. The first sign of recovery appeared to be an increased response to IPPB with Isuprel.

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VIII-B. COMMENTARY OF SELECTED ARTICLES

The conclusions of the U.S. Public Health Service Publications have been accepted by several organizations, government bodies and authors. Quotations from the following articles have been selected to illustrate the manner by which the conclusions have been quoted for special purposes. The articles are arranged according to the year of their publication.

Page			Reprint
162	(8)	FIESER L F: More on smoking and health. Chemistry 19: , 1964	817
164	(9)	KAMPMEIER R H: Smoking and noncancerous pulmonary disease. Southern Med J 57:981-3, 1964.	818
165	(10)	EDITORIAL. West Virginia Med J 60:51-2, 1964	819
166	(11)	RAVENHOLT R T: Cigarette smoking: magnitude of the hazard. Am J Public Health 54:1923-26, 1964.	820
167	(12)	WAKERLIN G E: Cigarette smoking and the role of the physician. Circulation 29:651-6, 1964.	821
168	(13)	FISHBEIM M: Medical progress in 1964.	822
169	(14)	U.S. DEPARTMENT OF HEALTH, EDUCATION AND WELFARE. Three kinds of evidence. Public Health Service Publication 1103-F:1-2, 1965.	823
171	(15)	FOGARTY J E: Delivered to the Task Force on chronic bronchitis and emphysema. NTA Bulletin 3-6, 1967.	824
172	(16)	SHAMBAUGH G E: Additional data on smoking. Arch Otolaryng 85: 589-90, 1967.	825
173	(17)	WHATLEY J L: Battle for breath. Today's Health 42-71, 1967.	826
174	(18)	ABELSON P H: Changing attitudes toward smoking. Science 161: 319, 1968.	827
175	(19)	DELARUE N C: Health hazards of smoking. Modern Med Canada 23:37-42, 1968.	828
176	(20)	HARKEN D E: Smoking . . . The Masochistic massacre. Dis Chest 54: 445-9, 1968.	829
177	(21)	HUTCHISON G.B: The nature of epidemiologic evidence: smoking and health. Bull NY Acad Med 44:1471-5, 1968.	830
178	(22)	NYLAN L H: Air pollution and emphysema. Connecticut Med 32:706 -7, 1968	831
179	(23)	OCHSNER A: Prevention of executive obsolescence. J Am Geriatrics Soc 16: 1077-82, 1968.	832

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Source: https://www.industrydocuments.ucsf.edu/docs/zmnk0000

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181	(25)	JACOBS E C: Smoking - insidious suicide and personal air pollution. Military Med 135:678-81, 1970.	834
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Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(8) FIESER L F : More on smoking and health. Chemistry 19: , 1964.

You may have seen headlines about the report of the Surgeon General's Advisory Committee on Smoking and Health (Chemistry, February 1964, page 2), but I doubt that you have waded through the 387-page document or the lengthy newspaper accounts of its findings. Since I was the only chemist on the 10-man advisory committee, I could hardly decline a request from the editor of Chemistry for a summary of the report addressed to Chemistry's readers. You may care to know what it all boils down to, so that you can make a wise decision for yourself and so that you can offer sound advice to others.

Countless weekend committee meetings were held in Washington in which we worked over a Friday-Saturday-Sunday stretch, continuing each day from 8:30 a.m. to about 8 p.m. One problem was that the medical men, who properly predominated on the committee, would not use a simple term such as "cancer of the mouth" when the more dignified medical term "carcinoma of the buccal cavity" was available.

The unanimous opinion of our committee was: "Cigaret smoking is a health hazard of sufficient importance in the United States to warrant appropriate remedial action."

A few of the findings on which this judgement was based are as follows:

- *Cigaret smoking is causally related to lung cancer in men; the magnitude of the effect of cigaret smoking far outweighs all other factors. The data for women, though less extensive, point in the same direction.*
- *The risk of developing lung cancer increases with duration of smoking and the number of cigarets smoked per day and is diminished by discontinuing smoking.*
- *Cigaret smoking is the most important of the causes of chronic bronchitis and increases the risk of dying from chronic bronchitis and from pulmonary emphysema.*
- *Male cigaret smokers have a higher death rate from coronary artery disease than nonsmoking males.*
- *The death rate for smokers of cigarets is about 70% higher than that for nonsmokers.*

These are strong words and they call for strong actions. It is not yet clear what remedial actions will be instituted by governmental agencies, but there is no question about the course young people should take. Don't smoke cigarets. Don't let yourself fall into the habit of smoking. It is a habit which many people regret but find difficult to break. You young people are in a position to make a decision in advance and so save yourselves from concern about remedial measures at a later, critical stage.

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Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

Commentary (8) Fieser, Cont.

The author of this Editorial has combined the two pulmonary diseases to read "increases the risk of dying from chronic bronchitis and from pulmonary emphysema." In the 1964 statement, the two diseases were covered in separate sentences: "cigarette smoking is the most important of the causes of chronic bronchitis" and "a relationship exists between pulmonary emphysema and cigarette smoking but it has not been established that the relationship is causal."

1005051480

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(9) KAMPMEIER R H: Smoking and noncancerous pulmonary disease. Southern Med J 57: 981-3, 1964.

Though there have been numerous demonstrations of impairment of pulmonary function in heavy cigarette smokers as compared to control groups, the main evidence of the hazard of cigarette smoking in terms of chronic bronchitis and eventual emphysema comes from epidemiologic studies. Everyone recognizes that air pollution, in cities especially and in certain occupations, can not be divorced from inhaled cigarette smoke when considering the ill effects of inspired air. Yet, other than asthma and familial tendencies to emphysema, chronic cough and infection are accepted as important factors in the development of emphysema. No one denies the "smokers' cough" and few would deny the airway resistance which is part of inhaled smoke, both unquestioned potentials in the development of emphysema. The pathologic evidence of alterations in the bronchial mucosa in prolonged cigarette smoking is so overwhelming that few argue about this anymore. The symptoms of this chronic bronchitis as a role in emphysema need no more than a mention.

What it all adds up to, is that no one has been able to show unquestioned causal relationship between cigarette smoking and chronic bronchitis and/or emphysema even approaching that of its relationship to bronchogenic carcinoma. But no matter how good a case the apologist for tobacco may make for chronic infection and air pollutants other than cigarette smoke as a cause for bronchitis and for emphysema, the fact remains that cigarette smoke is a pollutant which in its own right can impair pulmonary function and therefore has a deleterious effect on pulmonary structures over and above the other unavoidable factors in the air of our environment and the bacterial flora of our respiratory tract. It would be rather nonsensical to discard as accidental increased mortality rates in heavy cigarette smokers over those of control groups of nonsmokers in epidemiologic studies of chronic bronchitis and emphysema.

The application of epidemiologic studies to pulmonary emphysema and chronic bronchitis is mixed up with pathologic evidence. The prevalence of respiratory symptoms does not refer to incidence of these two separate disease entities. Although the 1964 statement has made a distinction between the influence of cigarette smoking to symptoms of bronchitis, others have interpreted the symptoms as indicating the positive presence of the disease.

1005051481

Source: https://www.industrydocuments.ucsf.edu/docs/zfnk0000

(10) EDITORIAL. West Virginia Med J 60: 51-2, 1964.

> An association between cigarette smoking and chronic lung disease has been established beyond medical argument. Cigarette smoking accounts for the majority of cases of emphysema and emphysema deaths now outnumber tuberculosis deaths. It has been estimated that smoking ten cigarettes per day will lead to a diminution of four years in life expectancy for the average American. Cancer of the lung is from 20 to 30 times more prevalent in the cigarette smoker than the nonsmoker. This is our most deadly cancer with a salvage rate of only 5 per cent. A study of British physicians shows that at age 35 the chances of dying within the next ten years are 1-23 for the heavy cigarette smoker and 1-90 for the nonsmoker.

The opening sentence refers to association between cigarette smoking and chronic lung disease but the second setence condemns smoking because it accounts for the majority of cases of emphysema. A causal relationship between cigarette smoking and emphysema has not been established, according to the 1964 report.

1005051482

Source: https://www.industrydocuments.ucsf.edu/docs/zmk0000

(11) RAVENHOLT R T : Cigarette smoking: magnitude of the hazard. Am J Public Health 54: 1923-26, 1964.

The censensus of findings of many studies[9] indicate that approximately nine-tenths of lung cancer deaths in the United States today are caused by smoking; and this consistent fact may be used in conjunction with the above findings (Table 1) to achieve a rough measure of total tobacco disease mortality:

Deaths from lung cancer in total population	122	1
Excess deaths from all causes among smokers	723	6

Hence, excess mortality from all causes among smokers in an American-type population is probably about six times greater than total lung cancer mortality in that population.

During 1962, 41,376 deaths were ascribed to lung cancer in the United States[10]; and by application of the lung cancer index of one-sixth, one can estimate that there were roughly a quarter million excess deaths due to smoking—which was 14 per cent of all deaths (1,756,720) from all causes. The relative importance of tobacco (especially cigarettes) as a hazard to life in the United States is indicated by the following balance of causes of death. (Figure 1.) The sum of all deaths from *accidents, infection, suicide, homicide, alcohol,* and *stomach cancer* only equals the quarter-million deaths from tobacco disease in the United States during 1962.

American Balance of Hazards to Life, 1962

Quarter Million Deaths from Tobacco	
*Lung Cancer	37,000
†Other Cancer Deaths (Of larynx, bladder, etc.)	23,000
‡Coronary Disease	119,000
‡Other Vascular Disease (Strokes, etc.)	35,000
‡Other Tobacco Diseases (Emphysema, bronchitis, etc.)	36,000

Quarter Million Deaths from Selected Causes	
All Accidents	97,139
Motor vehicle	40,804
Aircraft	1,254
Drowning	5,161
Other accidents	49,920
All Infectious Diseases	93,745
Tuberculosis	9,506
Pneumonia	66,999
Typhoid	15
Diphtheria	41
Other Infections	17,184
Suicide	20,207
Homicide	9,013
Alcohol	9,959
Stomach Cancer	19,378

* One-tenth of all (41,376) lung cancer deaths. † Number estimated from lung cancer index. (Total coronary disease deaths—327,581; total deaths from all causes—1,756,720.)

This article illustrates how portions of the 1964 statement has been used to equalize "excess death among smokers" with hazards to life. The death rate from emphysema and bronchitis is multiplied by lung cancer index and those compared with deaths from selected causes.

100505483

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(21) HUTCHISON G B : The nature of epidemiologic evidence: smoking and health. Bull NY Acad Med 44: 1471-5, 1968.

Epidemiologic Evidence

An extensive literature on associations between smoking and cardiovascular disease is now available. I propose to comment on some of the studies in this literature in which the unit of observation is a person and in which information is collected to characterize persons as to state of health. These are the studies which are commonly referred to as epidemiologic studies. When these studies are interpreted in terms of cause-effect relations the cause-effect concept is usually the pragmatic one.

The proof of cause-effect would be established or rejected by deliberately manipulating the smoking habits of such persons and observing the results. No such studies have been made to my knowledge. Controversy, when it occurs, rests on differences of opinion as to what constitutes proof in the absence of such a manipulative study. There are those who feel that proof is *never* established without such human experimentation. Others feel that proof can be established through observational studies, but there is little agreement as to what sorts of observations are required.

A number of schemes have been suggested for assuring such proof. Koch's postulates are an early attempt, but are applicable only when the causative event is exposure or nonexposure to living organisms. The surgeon general's committee has proposed a number of conditions which must be satisfied. These include consistency, strength, specificity, and coherence of the association. The ultimate decision, however, is said to be judgmental.

1005051484

The discussion of epidemiology is followed by the conclusion that the evidence is adequate to support the cause and effect relationship between lung cancer and cigarette smoking. If the same principle is applied to pulmonary emphysema, a cause and effect relationship has not been established.

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(22) NYLAN L H : Air pollution and emphysema: Connecticut Med 32: 706-7, 1968.

About 20,000 Americans will die of emphysema this year, seven times the number that succumbed to it a decade ago. It disables one out of fourteen American workers over forty-five years of age. E. Cyler Hammond,[1] Vice President of the American Cancer Society told a senate subcommittee that a recent study of 120,000 men indicates that a combined atmosphere of cigarette smoke and occupational pollution creates heightened risk of emphysema. A nonsmoker exposed to occupational pollution (smog, pollen, dust, exhaust fumes or other irritants) has one chance in ninety of developing emphysema. Among heavy smokers exposed to the same air pollution, one in seven develops this crippling disease. These findings are not new and are supported by those of others that air pollution and cigarette smoking often leads to destruction of lung tissue that characterizes emphysema.

The risk of smoker exposed to air pollution cited by Hammond in his testimony is one of 10 possible figures. The other reports do not indicate such a disparity between smokers and nonsmokers.

1005051485

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(23) OCHSNER A : Prevention of executive obsolescence. J Am Geriatrics Soc 16: 1077-82, 1968.

> Severe emphysema is a condition that can be considered worse than cancer. The patient with cancer of the lung either dies (only about 6 per cent can be cured) or gets well, whereas the patient with severe emphysema has a living death. According to the United States Public Health statistics, more than 12 million people in the United States today are completely disabled by severe emphysema caused directly by cigarette smoking. In 1965 in the United States, there were 77 million man-days lost from work because of tobacco, an additional 88 million man-days of sickness in bed as the result of use of tobacco, and 360,000,000 man-days of restricted activity because of the effects of tobacco. On the basis of these statistics, and using minimal figures of $40 a day for those totally disabled and $20 a day for those partially disabled, the loss of income from disability caused by tobacco in 1965 amounted to $15,800,000,000. The only reason that tobacco does not cause senescence more often is that frequently it causes early death—the dubious advantage of being spared early obsolescence.

The Surgeon General's report has been used to discuss tobacco and senescence. The author, a foremost surgeon, has accepted all the statistics and figures in 1967 document.

1005051486

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(24) SCHUMAN L M : Epidemiology of smoking related diseases which physicians encounter in their office practice. Dis Chest 54: 176-9, 1968.

Chronic bronchopulmonary diseases were cau
ly implicated in the 1964 Advisory Committee
port to the Surgeon General. The evidence v
deemed adequate for a causal relationship betw
cigarette smoking and chronic bronchitis and
was strongly implicated as increasing the risk
death from pulmonary emphysema. Despite
recent attempts at more precise definitions of chr
ic bronchitis and pulmonary emphysema, the sta
of such definitions over the immediate past in
own country and those in other countries mak
trends and comparisons difficult if we attempt
separate the two entities. Thus, mortality and m
bility analyses are better applied to a combination
of the two—chronic bronchitis and/or emphysema.
As a problem in *mortality*, chronic bronchitis and/
or emphysema presently claim half as many deaths
as does lung cancer. However, in the 16-year peri-
od, 1950–65, deaths from these two non-neoplastic
pulmonary diseases have increased over six-fold
from 3,157 deaths reported in 1950 to 23,432 re-
ported in 1965.[4,5] This increase has been greater
than the similarly appalling toll from lung cancer.
The most dramatic rise has been for the emphy-
sema component for which the age-adjusted mor-
tality rates rose from 1.3 per 100,000 in 1950 to al-
most 13.0 in 1965. Deaths from chronic bronchitis
rose less spectacularly, only doubling in the same
period. Although some of these increases must
represent diagnostic improvement, it is difficult to
ascertain the degree to which this operated. The
causal relationship with cigarette smoking and the
use of cigarettes in this period would suggest that
a major part of this rise in mortality must be real.

The explanation for combination of chronic bronchitis and emphysema in mortality statistics is not a convincing one. Since 1964, the diagnosis of the two disease entities has advanced to the point of widespread application. It would be reasonable to analyze the death rates from the two diseases separately.

100505487

Source: https://www.industrydocuments.ucsf.edu/docs/zjmk0000

(12) WAKERLIN G E : Cigarette smoking and the role of the physician. Circulation 29: 651-6, 1964.

Smoking and Other Diseases

Epidemiologic, experimental, and clinical studies have demonstrated adverse relationships between cigarette smoking and lung cancer, chronic bronchitis, and emphysema. One of the complications of emphysema is cor pulmonale. Cigarette smoking has also been shown to be associated with an increased incidence of arteriosclerosis of the pulmonary arterioles and small arteries.

Increased morbidity and mortality among cigarette smokers have been reported for certain other forms of cancer and for peptic ulcer.

Smoking and Over-all Death Rates

Cigarette smokers show adverse changes in structure and function of the lungs and bronchial tree.[18, 19] Pulmonary function changes include decreased vital capacity and decreased forced expiratory volume.[20] Some of these changes revert toward normal in those who stop smoking.[21]

In addition to the greater risk of illness and death from disease, recent studies suggest a reduced sense of well-being among cigarette smokers. Thus, compared with absence of the cigarette habit, cigarette smoking is more frequently associated with cough, shortness of breath, decreased appetite, and increased "nervous tension."[22] An adverse association of cigarette smoking with body function appears to begin with fetal life, since more premature infants are born to mothers who smoke cigarettes.[23, 24]

Recent Actions with Reference to Smoking

10050514488

The statements are individually accurate. The implication to the reader is unavoidable: pulmonary effects of cigarette smoking is responsible for deaths which in turn has led to recent actions against smoking.

Source: https://www.industrydocuments.ucsf.edu/docs/zmk0000

(13) FISHBEIM M : Medical progress in 1964.

> The rise in cancer of the lung was associated with a considerable increase in emphysema in many parts of the world, particularly in the U.S., Canada, and England. One survey indicated that about 25% of men over 45 have some degree of emphysema. The condition is from seven to 13 times more frequent among cigarette smokers than among nonsmokers. Both cigarettes and air pollution have been considered as causative factors. Great attention is now focused on determining the exact relationships among these factors and diseases.

> Looking back at 1964, a few peaks of interest become apparent in each area of medical research and medical practice. Heart disease, cancer, and high blood pressure with stroke received major medical attention as the leading causes of death. Infectious diseases were controllable as never before, and more of the infectious diseases of childhood became wholly amenable to prevention and cure.

The survey showing that 25% of men over 45 have some degree of emphysema refers to prevalence of coughing and expectoration. The quoted 7 to 13 times more frequent among cigarette smokers is also based on incidence of respiratory symptoms. The mortality ratio for pulmonary emphysema among smokers compared to nonsmokers is about 3 times.

1005051489

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(14) U. S. Department of Health, Education and Welfare. Public Health Service Publication 1103-F: 1-2, 1965.

SURGEON GENERAL'S COMMITTEE

By 1962 scientific research had implicated the cigarette in several major diseases. In addition to chronic bronchitis and emphysema, these included heart disease and lung cancer. That year, the Surgeon General of the Public Health Service appointed an advisory committee of 10 eminent scientists to review the accumulated evidence. The committee had advice and consultation from nearly 200 specialists representing every related field of interest.

THREE KINDS OF EVIDENCE

The Surgeon General's Committee reviewed three kinds of research evidence.

1. Animal Experiments

Animals exposed to tobacco smoke show severe damage to the cells lining the air tubes—the same kind of damage seen in heavy smokers. (A pack a day or more is considered "heavy smoking.")

2. Human Tissue

When the cells of the bronchi of smokers and nonsmokers are compared, the smokers' cells show characteristic damage: loss of the cilia and irregular shapes. These changes have been noted in thousands of studies, both in living patients and at autopsy.

3. Population Studies

Extensive and repeated studies of large numbers of people have consistently shown that cigarette smokers are more likely to have symptoms of respiratory disease than nonsmokers. They cough more, bring up more phlegm, and complain more of breathlessness.

SMOKING AND HEALTH REPORT

In January 1964, the Surgeon General's Committee finished the most comprehensive study of smoking and health ever undertaken. Studies involving more than a million men had been reviewed. For cigarette smokers, the death rate from chronic bronchitis and emphysema was found to be *six times* greater than for nonsmokers. A 500-percent difference! Ex-smokers had a death rate from these diseases about 40 percent greater than for nonsmokers. Men who began smoking before the age 20 had a higher death rate than those who started later.

10050514 90

Source: https://www.industrydocuments.ucsf.edu/docs/zlak0000

Commentary (14) U.S. Department of Health, Education and Welfare, Cont.

This widely distributed leaflet contains selected statements, each one quoted from the 1964 report. The combination of the three kinds of evidence do not relate to chronic bronchitis or pulmonary emphysema mentioned above and below the quotation. The animals were not sick with emphysema or bronchitis, the human tissue was obtained from smokers who were not suffering from emphysema or bronchitis, and the population studies surveyed respiratory symptoms which do not necessarily signify emphysema or bronchitis.

1005051491

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(15) FOGARTY J E : Delivered to the Task Force on chronic bronchitis and emphysema. NTA Bulletin 3-6, 1967.

> What is the cause—or should I say causes—of emphysema, the condition that begins innocently enough with a mild cough and seems to proceed to increasing shortness of breath until the normal activities of the day become increasingly burdensome?
>
> Is cigarette smoking a factor? We are told in the report of the Surgeon General's Advisory Committee on Smoking and Health that cigarette smoking is an important cause of chronic bronchitis and that a "relationship" exists between cigarette smoking and emphysema. Has that relationship as yet been defined? Despite the accumulating evidence that cigarette smoking and emphysema are intimately linked, our society is heedlessly smoking six per cent more cigarettes a year than in 1964, when the report of the Surgeon General's committee was released.
>
> What about air pollution? In all honesty, we do not need experts to tell us that air pollution is an insult to our respiratory tract, yet until now our national complacency has permitted the continued spewing of poisons into the air.

The relationship of cigarette smoking to chronic bronchitis and pulmonary emphysema is differentiated in this article, according to the 1964 report. The term cause is unfortunate; risk factor would have been more appropriate.

1005051492

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(16) SHAMBAUGH G E : Additional data on smoking. Arch Otolaryng 85: 589-90, 1967.

THE NATIONAL Center for Health Statistics in October 1966 published the Mortality Statistics From Diseases Associated With Smoking, from 1950 to 1964. Two groups of diseases are considered: those *causally related* to smoking, namely cancer of the lung, cancer of the larynx, cancer of the lip, and chronic bronchitis, and those *associated* with smoking but where the evidence is not yet sufficient to establish a causal relationship. The latter group comprises arteriosclerotic heart disease including coronary disease, cirrhosis of the liver, emphysema, ulcer of the stomach, cancer of the esophagus, cancer of the oral cavity other than the lip, cancer of the bladder and other urinary organs, and noncoronary cardiovascular disease. *JAMA* (199:24 [Jan 23] 1967) carries an interesting study of cigarette smoking in dogs. After more than 420 days of smoking through a tracheostomy tube there was marked cardiac enlargement compared to controls, and pulmonary fibrosis and emphysema in all the smoking dogs, not found in any of the controls. Interestingly some of the dogs showed a liking of cigarette smoking as indicated by tail-wagging and jumping into the smoking box voluntarily. With this evidence perhaps emphysema should be reclassified as causally related to smoking, rather than associated with smoking.

The widely publicized "smoking dogs" has undoubtedly led to the reversal of the statement from no causal to causal relationship between cigarette smoking and pulmonary emphysema. There are other attempts in animals which failed to show a cause and effect relationship but these were never publicized.

100505493

Source: https://www.industrydocuments.ucsf.edu/docs/zmk0000

(17) WHATLEY J L : Battle for breath. Today's Health 42-71, 1967.

Emphysema and chronic bronchitis are the fastest rising causes of death in the country. Almost 20,000 people will die this year from emphysema or its complications. Whether this increase is due to better methods of diagnosis, or to the greater longevity of our population, we do not know. Certainly, part of the increase is due to modern environmental conditions (exhaust fumes, smog, cigarette smoke, etc.).

Emphysema is about 13 times more prevalent among cigarette smokers than among non-smokers, but many who have *never* smoked contract the disease. Men develop emphysema five to 10 times more frequently than women, probably because women are "abdominal breathers" (they use the diaphragm and abdominal muscles more), whereas men are "chest breathers" (they use the chest muscles more).

At present, we don't know the exact cause of pulmonary emphysema, but we do know quite a bit about it. We know that it is an overinflation of the lung due to loss of that organ's natural elasticity. The smaller airways (bronchial tubes) narrow from spasm, irritation, infection, or a combination of the three. This narrowing traps the air in the small air sacs (alveoli), and they dilate and lose their elasticity, or ability to recoil.

The 13 times prevalence of emphysema among cigarette smokers is based on incidence of respiratory symptoms which does not necessarily signify the disease. The author avoids stating that emphysema is caused by cigarette smoking. Even though the author does not know the exact cause of pulmonary emphysema, the hurried reader will undoubtedly regard the 13 times prevalence of cigarette smoking as a causative factor.

1005051494

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(18) ABELSON P H : Changing attitudes toward smoking. Science 161: 319, 1968.

The attitudes of the people of the United States toward cigarette smoking are undergoing a major change. In the 30-year period prior to 1964, per capita consumption of cigarettes tripled. During the last 4 years, per capita consumption of cigarettes has leveled off, and currently it may be dropping. The 1964 Surgeon General's Advisory Committee report entitled "Smoking and Health" was a major milestone. The report set in motion developments that ultimately will have a profound effect. One consequence was the stimulation of research bearing upon the effects of smoking. A recent compendium lists 364 projects in 36 states, the District of Columbia, and 25 foreign countries.

Out of this research activity has come substantial evidence that the effects of smoking are even more serious than was stated in the 1964 report. Highlights of the findings have been presented in a new U.S. Public Health Service document.* The most striking finding is that "the life expectancy for a two-pack a day, or more, smoker at age 25 is 8.3 years less than [that for] the corresponding non-smoker."

The 1964 report probably has influenced the smoking habits of physicians. At one time many of them were heavy smokers. A recent survey† indicates that 100,000 physicians have stopped smoking cigarettes. Of all the physicians surveyed, 35 percent had never smoked, 36 percent had smoked at one time but had stopped, and only 29 percent were still smoking. In contrast, 52 percent of the general adult male population currently smoke cigarettes.

The survey also indicates that physicians are deeply concerned over the effects of smoking. More than 90 percent stated that they associate smoking with chronic bronchitis, lung cancer, and emphysema, and almost as many associate it with peripheral vascular disease and coronary artery disease. The responses of physicians who have stopped smoking are revealing. When asked on the survey questionnaire why they stopped, 60 percent checked "Protect my future health," 47 percent checked "Occurrence of certain symptoms," and 43 percent checked "Scientific reports convinced me."

Following the appearance of 1967 Report, a second wave of Editorials appeared. The smoking habits of physicians are discussed here. It is important to point out that 43% who stopped smoking were convinced of the scientific reports summarized in the U. S. Public Health documents.

100505495

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(19) DELARUE N C : Health hazards of smoking. Modern Med Canada 23: 37-42, 1968.

> 2. *Chronic Bronchitis and Emphysema.*
> There are now 80,000 annual deaths in the United States from these diseases and although they both may develop in non-smokers, the risk of death in cigarette smokers is increased from eight to 15 times as compared to the non-smoking population. In the five-year period between 1959 and 1964 deaths from chronic bronchitis increased 90 per cent and over the same period, deaths from emphysema increased 104 per cent. There were 421,000 new cases of bronchitis recorded in 1965, and 267,000 new cases of emphysema defined in the same period. At that time, it was estimated that there were more than three million cases of chronic bronchitis in the United States.

This special article appeared in a Canadian journal. It can be noted that United States statistics are quoted here, even though there is information directly from Canada. Other countries have relied on the U. S. Public Health documents for statistics relating to chronic bronchitis, although its incidence varies from country to country.

10050514 96

Source: https://www.industrydocuments.ucsf.edu/docs/zjmk0000

(20) HARKEN D E : Smoking . . . The Masochistic massacre. Dis Chest 54: 445-9, 1968.

The combination of seven prospective studies reveals that the mortality rates of cigarette smokers over non-smokers for all causes of death was 7 per cent higher in smokers.

Let us look at some cold facts on mortality in a combination of seven prospective mortality records of smokers versus non-smokers:

Cancer of lung	(10.8)	1000 per cent (leading cancer in men)
Bronchitis and emphysema	(6.1)	600 per cent (and leading cause of disability)
Cancer of the larynx	(5.4)	
Oral cancer	(4.1)	
Cancer of the esophagus	(3.4)	
Peptic ulcer	(2.7)	
Atherosclerotic disease	(2.6)	
Coronary artery disease	(1.7)	70 per cent

Although death rates of chronic bronchitis separately from pulmonary emphysema are available, the 1967 report combines the two diseases, presumably to improve the statistical significance. Reference to the mortality ratio for smokers to nonsmokers appearing in articles such as the one under consideration do not question the practice.

10050514 97

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(25) JACOBS E C : Smoking: insidious suicide and personal air pollution. Military Med 135: 678-81, 1970.

Facts

Smoking remains the foremost preventable cause of disease, disability and death in the United States.[8-13] Smoking causes coronary artery disease, lung cancer, emphysema, thrombo-angiitis obliterans and the like, annually producing hundreds of thousands of premature deaths and many hundreds of thousands of cripples. Tobacco diseases are causing many millions of man-days lost annually in this country. Tobacco diseases can shorten the smoker's life by eight or more years,[12] can cause a death every 1.5 minutes,[15] and cost the Nation between $4 billion[9] and $20 billion annually.[13]

The statements in the U. S. Public Health documents are abbreviated in this short paragraph. It can be noted that smoking is stated to cause pulmonary emphysema. The phrase "no causal relationship has not been established" has been omitted.

10050514998

Source: https://www.industrydocuments.ucsf.edu/docs/zfmk0000

(26) BENNETT J S : Smoking: time to practice what we preach. Canad Med Assoc J 106: 276, 278 1972.

The 1962 report on Smoking and Health by the Royal College of Physicians (London) was discounted by the tobacco industry as contributing little but "statistical association". The College's new report "Smoking and Health Now" (1971) points out that the criticism of "statistical evidence only" cannot be upheld as a valid objection. Much of aetiology, morbidity incidence, etc., on human disease is based on statistical evidence; the pro-smoking faction suggests that, as far as smoking is concerned, no laboratory evidence is available to show its responsibility for lung cancer. However, the recent work of Auerbach and Hammond on invasive lung cancer in dogs should convince the Doubting Thomas. Statements from the U.S. Department of Health, Education and Welfare (1971) are:

1. Cigarette smoking is the major cause of lung cancer.
2. Cigarette smoking is the most important cause of chronic obstructive broncho-pulmonary disease, principally chronic bronchitis and pulmonary emphysema.
3. Cigarette smoking is an important risk factor in the development of coronary heart disease and, by accelerating damage already present as a result of coronary heart disease, may contribute to sudden death.

Isn't it time that the medical profession became a little more active in the efforts to reduce one of the major health hazards of our time. We tend to get worked up about pollution but do little about one of the greatest contaminants of all — the tobacco smoker.

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The 1971 U. S. Public Health document and the 1971 report from the Royal College of Physicians are cited in this article. The widely publicized smoking dogs are cited as the most important laboratory evidence to support the statement that cigarette smoking is the major cause of lung cancer and the most important cause of chronic bronchitis and pulmonary emphysema.

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VIII-C. PUBLICITY RELATING TO SMOKING AND HEALTH

The widespread distribution of the seven documents on "Smoking and Health" has led to the appearance of secondary publications. Almost all of them accept the conclusions appearing in the seven documents that cigarette smoking is the most important cause of chronic bronchitis and pulmonary emphysema. A few articles contest the statement but none has appeared in recent years. The articles are listed in the following order:

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C-(1).	Articles and publicity favoring Surgeon General's statement. (total number - 62)	184
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IX. RECOMMENDATIONS

The reviewer is submitting the following recommendations to the Council for Tobacco Research:

1. Publication. That cigarette smoking is one of several risk factors and not the most important cause of chronic pulmonary disease should be publicized to the medical profession and the general public. Portions of this review can be expanded and modified for publication and distribution.

2. Epidemiology. The expert advice of a statistician is needed to review mortality statistics, specifically to analyze the death rate for pulmonary emphysema separately from that for chronic bronchitis. The morbidity statistics should also be examined for regional differences in the United States and comparison between countries.

3. Clinical investigation. A five-year follow-up of smokers should be conducted to determine whether those with early signs of ventilatory function develop pulmonary emphysema or chronic bronchitis.

4. Pathological investigation. A registry of persons who have been smokers for 50 years would be helpful in determining the influence of cigarette smoking.

5. Animal investigation. Attempts to induce pulmonary emphysema or chronic bronchitis in animals should continue. The emphasis should be given to determining the relative importance of air pollution, infection and cigarette smoking in the same laboratory model.

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X. ADDITIONAL BIBLIOGRAPHIC LISTS

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